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An Overview on Sudden Cardiac Death

Bibin Rijo W, Aswin A V, Divika S, Dr. K C Arul Prakasam, Dr. N Senthil Kumar

Department of Pharmacy Practice, Annai JKK Sampoorani
Ammal College of Pharmacy, Komarapalayam, Tamil Nadu, India

ABSTRACT

As the most prevalent cause of death in developing nations is heart disease, over 7 lakh people in India and over 4-5 million worldwide pass away from sudden cardiac death (SCD) each year. It is the most prevailing kind of unexpected mortality brought on by cardiac anomalies like congenital heart disorders, hereditary channelopathies, heart failure and ischemic heart diseases. Nevertheless, non-cardiac causes such aortic syndromes, stroke and pulmonary embolism can also result in sudden cardiac death and must to be taken into account as alternative diseases. Additionally, younger individuals experience sudden cardiac death, which is pertaining to obesity, stress, lifestyle changes, alcoholism and fibrosis (non-ischemic causes of sudden cardiac death). This study exemplifies the causes of sudden cardiac death (SCD), most notably for those with cardiovascular diseases.

KEYWORDS: *Sudden Cardiac Death; Ischemic Heart Disease; Inherited Channelopathies; Cardiomyopathies; Heart Failure*

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INTRODUCTION

Heart disease is major cause of death in developing countries like India [1]. Sudden cardiac death (SCD), the most common type of sudden death, is caused by cardiac abnormalities [2]. Sudden cardiac death (SCD) is an unexpected sudden death due to a heart condition, which is almost always confirmed upon post-mortem examination [3]. However, non- cardiac causes such as pulmonary embolism, stroke, and aortic syndromes can also lead to rapid death and should be considered as alternate pathologies [4]. Survival after SCA remains very low stable (approximately 7%), despite major investments by the medical and research communities in this area over the past decades[5]. It is estimated that there are about 4-5 million incidents of sudden cardiac death worldwide each year. It can be estimated that over 7 lakh SCD cases occur in India each year[5]. The causes of death vary with the age of the patient. A cardiac cause of sudden infant death syndrome has yet to be established [8]. SCD, however, can also happen in young people when an underlying hereditary or congenital disease directly affects the myocardium electrical system of the heart [9]. Arrhythmic factors are likely the primary cause of sudden cardiac death in individuals under the ages of 35 [10]. About 80% of SCD is caused by coronary artery disease. Cardiomyopathies and genetic channelopathies account for the remaining causes. Cardiomyopathy associated with obesity, alcoholism and fibrosis are causes of non-ischemic sudden cardiac death [11].

ISCHEMIC HEART DISEASE:

A. MYOCARDIAL INFARCTION

Patient with underlying coronary artery disease account for the great majority of SCD cases, with the risk high in patients who have suffered an MI [12]. Sudden death occurs frequently during the acute phase of MI as a result of ischemia, which causes fatal ventricular arrhythmias [13]. In between 25% and 50% of patients with a history of MI, sudden death, is most frequently brought on by ventricular tachycardia (VT) or ventricular fibrillation (VF). ICDs (Implantable cardioverter-defibrillators), can greatly lower the incidence of arrhythmic sudden death in MI patients [14]. The Canadian implantable defibrillator study (CIDS) and the Hamburg Cardiac Arrest Study (CASH) have both demonstrated that

ICDs can increase patient survival and decrease mortality in people with deadly ventricular tachyarrhythmia (VTA). Mortality increase when LVEF (Left Ventricular Ejection Fraction) declines< 50% due to improvement in rapid reperfusion therapy during acute MI, relatively few patients has very low LVEF after MI [16].

B. ANOMALOUS CORONARY ORIGIN

The most frequent life threatening anomaly linked to a higher risk of SCD is an anomalous origin of a CA from the contralateral sinus of Val Sava, also known as an anomalous aortic origin of CA [17]. Anomalous aortic origin of a coronary artery is the second leading cause of sudden leading cause of sudden cardiac death (SCD) in young US athletes. The symptoms that patients with AAOCA (Anomalous aortic origin of the coronary artery) present with are highly diverse, ranging from evident myocardial ischemia to sudden cardiac arrest (SCA)[18]. Isolated unroofing is the method used most frequently to treat AAOCA in children and adolescents. Depending on the type AAOCA reimplantation pulmonary artery translocation, patch augmentation, and hybrid combination approaches are the other reported anatomic techniques [19].

C. CORONARY SPASM :

Ischemic heart disease, various forms of angina, acute myocardial infarction, and sudden cardiac death, is caused by coronary artery spasm. Coronary artery spasm is an abnormal contraction of an epicardial coronary artery [20]. The pathophysiology of spasms in diseased coronary arteries may be caused by impaired Nitric Oxide (NO) generation in diseased segments, a lack of sufficient response, as well as other variables. Products made from platelets may have a major role in the whole interaction, worsening the spasm [21]. Medical professionals employ an angiography to get an X-ray image of the heart arteries while administering acetylcholine injections, which are intended to relax blood vessels, coronary artery spasms can be identified if the blood vessels contracts instead (vasospasm) [22]. The oral spray of nitroglycerin or isosorbide dinitrate (ISDN) or sublingual administration of nitroglycerin can typically quickly ease a incident of coronary spasm. Intravenous or intracoronary may be required for refractory spasm [23].

INHERITED CHANNELOPATHIES :

A. Long QT syndrome

Long QT syndrome (LQTS) is an inherited primary arrhythmia disease that can cause malignant arrhythmia and, in risk of sudden death [24]. The depolarization and repolarization phases of the cardiac action potential are represented by the electrocardiographic QT interval. The length of the action potential is governed by the interactions of numerous ion channels. A pathophysiological substrate for LQTS is a decrease in repolarizing outward potassium currents or an increase in depolarizing inward sodium or calcium currents, both of which can lengthen the QT interval [25]. The majority of people with LQTS have an affected parent. Beta-blockers medication is the primary treatment for LQTS. For patients with high risk, ICD therapy is considered prophylactic[26].

B. Short QT syndrome:

A rare form of inheritable cardiac channelopathy known as short QT syndrome (SQTS) is distinguished by abnormally short QT intervals on the ECG [27]. Short QT syndrome has an increased risk of familial atrial fibrillation and/ or sudden cardiac death [28]. The symptoms of short QT syndrome are palpitations, atrial fibrillation, syncope, and SCD [29]. Diagnosis of short QT interval was based upon an ECG with QT intervals< 300 ms at a normal heart rate. Hydro quinidine is used in the treatment of short QT syndrome and ICD is implanted for primary and secondary prevention of SCD [30].

C. Brugada syndrome

Brugada syndrome (BrS) is a very rare inherited arrhythmia condition that is associated with ventricular fibrillation (VF) and sudden cardiac death (SCD) but has no evident structural abnormalities [31]. Brugadasyndrome (BrS) is characterized by ST-segment elevation in the right precordial ECG leads [32]. Patients with a higher risk can be identified by a spontaneous type 1 ECG, inducible VAs during an EPS (Electrophysiological study), and the presence of SND (Sinus Node Dysfunction) [33]. Patients with BrS may have palpitations, syncope or nocturnal agonal breathing, which could lead to ventricular arrhythmias or SCD [34]. Implantable cardioverter-defibrillators (ICD) are the only proven effective method for preventing SCD in BrS patients. Currently used pharmacological therapies include quinidine and phosphodiesterase III inhibitors [35].

D. Early repolarization syndrome

In early repolarization syndrome, a dispersion of de- and repolarization is caused by current imbalances between the epi- and endocardial layers. On the surface ECG, these imbalances appear as J waves or elevated ST segments [36]. The junction between the start of the ST segment and end of the QRS complex is known as J point [37]. Recent studies have suggested a link between ER and a higher risk of cardiac arrhythmia-related death [38]. The implantation of an implanted cardioverter defibrillator is advised in individuals with an early repolarization

pattern who survived sudden cardiac death. A continuous infusion of isoproterenol prevents recurrent ventricular fibrillation. Quinidine and hydro quinidine have been described as effective treatment for early repolarization syndrome's ventricular fibrillation and ventricular tachycardia [36].

E. Catecholamiogergic Polymorphic Venricular Tachycardia (CPVT):

The channelopathy known as catecholaminergic polymorphic ventricular tachycardia (CPVT) does not have apparent structural heart disease. It is a heterogeneous disease characterized by a normal resting ECG, ventricular events brought on by exercise, acute emotion, stress-induced or adrenaline mediated premature ventricular contractions (PVCs), polymorphic ventricular tachycardia (PVT), and/or bidirectional VT (BVT), with recurrent nature and typically when the heart rate (HR) get above 120 bpm [39]. Autopsy-negative sudden death is significantly caused by CPVT. CPVT mainly affects children with a mean age of 7-9 years [40]. Using exercise stress testing, VA is most frequently shown to be present for the diagnosis of CPVT [41]. Non-selective beta-blockers are the initial drug treatment option that is most frequently used for CPVT. In most nations, nodolol (1-2 mg/kg per day)is the primary choice; nevertheless, proponolol (3-5 mg/kg per day) may be administered if nadolol is not accessible. It has been demonstrated that flecainide (100-300 mg daily) reduces the burden of arrhythmias. Left cardiac sympathetic denervation (LCSD) is a procedure that can be used on patients who are resistant to the most effective pharmacological treatment and it significantly reduces the number of arrhythmic episodes. For individuals who have not responded to maximal pharmacological treatment and LCSD, guidelines recommend an implanted cardiac defibrillator (ICD) [42].

CARDIOMYOPATHIES

Myocardial disorders in which in the heart muscle is structurally and functionally abnormal, and in which coronary artery disease, hypertension, valvular and congenital heart disease are absent or do not sufficiently explain the observed myocardial abnormality [43]. Leading causes of SCD have been identified as hereditary cardiomyopathies and cardiac channelopathies. Hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), restrictive cardiomyopathy (RCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), and left ventricular non compaction (LVNC) are the most prevalent SCD-related cardiomyopathies in young children and adults [44].

The initial diagnosis is made with an echocardiography test. To diagnose and treat dilated cardiomyopathy, the EKG must be conducted at the initial evaluation. Laboratory testing can provide etiological information. MRI, coronary angiography, endomyocardial biopsy, and genetic testing are also used to diagnose cardiomyopathy. The main causes of SCD related cardiomyopathies are;

- HYPERTROPIC CARDIOMYOPATHY
- RESTRICTIVE CARDIOMYOPATHY
- ARRHYTHMOGENIC RIGHT VENTRICULAR CARDIOMYOPATHY
- DILATED CARDIOMYOPATHY [45]

HEART FAILURE:

Heart failure (HF) is a syndrome defined by varying degrees of inadequate perfusion and congestion of the systemic and/or pulmonary venous systems [46]. Among the cardiovascular deaths, the most common cause of mortality in HFpEF was sudden death [47]. A third heart sound indicates an increase in left ventricular end-diastolic pressure and a decrease in LVEF. BNP and N- terminal pro-BNP levels can be used screen patients for heart failure who have dyspnea. Chest radiography and electrocardiography are used to diagnose heart failure [48]. Implantable cardioverter defribillators (ICDs) are at present only recommended as a main preventive therapy in selected patients with low ejection fraction [49]. ACE inhibitors, ARBS, ARNI'S, MRA'S, Beta blockers, nephrilising inhibitors, hydralazine/isosorbide dinitrate combination, ivabridine, digoxin, soluble guanylate cyclase stimulators, diuretics, SGLT2 inhibitors, calcitropes, myotropes, mitotropes are drugs used in the treatment of heart failure [50].

CONGENITAL DISEASE

A. Tetralogy of fallot

Tetralogy of Fallot(TOF) is the most common type of cyanotic type of cyanotic congenital heart disease. TOF patients exhibit varied degrees of cyanosis depends on the severity of right ventricular outflow tract RVOT stenosis and pulmonary artery (PA) anatomy [51]. Tetralogy of Fallot affects 3 out of every 10,000 live births. It is the most common cause of cyanotic cardiac disease in patients over the age of one, accounting for up to one-tenth of all congenital cardiac abnormalities [52]. Patients with tetralogy of fallot have SCD rates of 0.1-0.2%/ year [53]. Echocardiography, computed tomography angiography, or magnetic resonance imaging is used in the diagnosis of tetralogy of fallot. The standard for tetratology of fallot is surgical repair, which includes patch closure of the ventricular septal defect and extensive relief of right ventricular outflow tract

blockage. After the surgical repair is complete, cardiac catheterization can be used to treat residual lesions or long-term problems such as pulmonary artery stenosis, residual or additional ventricular septal defect, or pulmonary valve regurgitation. A stunt is placed to prevent blockage and monitor for complications [54].

CONCLUSION :

These days, Sudden cardiac death is most common. According to recent studies, 80% of cases of sudden cardiac death are attributable to coronary heart disease and lifestyle factors including alcoholism and sedentary behavior. Therefore, this article recommends routine monitoring, particularly for people with cardiac conditions.

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